

PubMed	Nucleotide	Protein	Genome	Structure	PMC	Taxonomy	OMIM	Books
Search PubMed	for systemic shock and marburg					Preview	Go	
Clear								
Limits		Preview/Index		History	Clipboard		Details	

- Search History will be lost after one hour of inactivity.
- To combine searches use # before search number, e.g., #2 AND #6.
- Search numbers may not be continuous; all searches are represented.

Entrez  
PubMed

Search	Most Recent Queries	Time	Result
#15	Search systemic shock and marburg	14:59:56	<u>5</u>
#14	Search systemic shock and ebola	14:59:45	<u>0</u>
#13	Search systemic shock and Lassa	14:59:34	<u>0</u>
#11	Search systemic shock and Dengue	14:58:33	<u>5</u>
#9	Search pulmonary distress and Dengue	14:57:57	<u>1</u>
#7	Search pulmonary distress and Lassa	14:57:23	<u>1</u>
#6	Search pulmonary distress and Lassa virus	14:57:17	<u>0</u>
#5	Search pulmonary distress and MARburg	14:56:25	<u>16</u>
#4	Search pulmonary distress and Ebola	14:55:48	<u>0</u>
#3	Search systemic shock and Ebola	14:55:23	<u>0</u>
#1	Search systemic shock and SNV	14:54:00	<u>1</u>

PubMed  
Services

Related  
Resources

Clear History

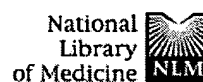
Write to the Help Desk  
NCBI | NLM | NIH  
Department of Health & Human Services  
Freedom of Information Act | Disclaimer

## WEST Search History

DATE: Wednesday, April 16, 2003

<u>Set Name</u> side by side	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u> result set
<i>DB=USPT; THES=ASSIGNEE; PLUR=YES; OP=ADJ.</i>			
L4	L3	491	L4
<i>DB=USPT,PGPB,JPAB,EPAB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ</i>			
L3	lymphotoxin and shock	935	L3
L2	L1 and lymphotoxin	9	L2
L1	browning J.in.	130	L1

END OF SEARCH HISTORY



PubMed	Nucleotide	Protein	Genome	Structure	PMC	Taxonomy	OMIM	Books
Search PubMed	for						Go	Clear
Limits Preview/Index History Clipboard Details								

Display Abstract	Show: 20	Sort	Send to Text
------------------	----------	------	--------------

☐ 1: Cancer Biother Radiopharm 1998 Jun;13(3):193-207

[Related Articles, Links](#)

Entrez  
PubMed

## Implications of the analogy between recombinant cytokine toxicities and manifestations of hantavirus infections.

Wimer BM.

PubMed  
Services

JBMW Immunotherapeutics, Albuquerque, NM 87123-4255, USA.

Related  
Resources

The etiologic hantavirus of the 1993 emergence of an acute pulmonary failure syndrome in the area around northwestern New Mexico was quickly recognized as related to the Hantaan virus responsible for the outbreak of Korean epidemic hemorrhagic fever (EHF) among UN troops in 1951. Discovery of the new disease which was named the hantavirus pulmonary syndrome (HPS) and its causative agent the Sine Nombre virus (SNV) inspired detailed comparisons between the two disorders. Major damage to the epithelial cells of the capillaries and arterioles throughout the body leading to extensive capillary leak and subsequent hypotension and shock was the common denominator. The lung capillaries and arterioles were the focus of attack that could lead to rapid pulmonary failure in HPS and the corresponding renal and retroperitoneal vessels that caused a more protracted illness in EHF, but both displayed remarkably similar peripheral blood abnormalities including abnormal mononuclear cells, immature neutrophilia, thrombocytopenia, and hemoconcentration characteristic enough to make blood smear examination a useful tool in early diagnosis. There are evidences that a heavy virus presence in the involved endothelial cells is accompanied by various mononuclear cells capable of generating potent immune response in these areas. Relevant toxic effects of systemically-administered high-dose interleukin-2 for resistant cancers include fever, chills, diarrhea, renal dysfunction, capillary leak syndrome accompanied by hypotension requiring aggressive pressor support, and occasional pleural effusions with diffuse pulmonary infiltrates and hypoxia severe enough to require ventilatory assistance. Peripheral blood mononuclear cells cultured in vitro with IL-2 secrete secondary cytokines such as IL-1, TNF-alpha, and interferon-gamma (IFN-gamma). TNF-alpha, implicated in the pathophysiology of septic shock, is capable of inducing adult respiratory distress syndrome (ARDS) in experimental animals and humans. The strong similarity of these effects to the manifestations noted in the hantavirus diseases justifies the conviction that these and other cytokines involved in potent immune responses would constitute the pathogenic toxic substances predicted by perceptive early investigators of EHF. This concept is favored by clear indications that in both diseases active virus infection disappears the first few days and the ages of involvement correlate with periods of immunocompetence. The paradox of

systemic injections of IL-2 that risk hantavirus-type toxicities for treating renal cell carcinoma and melanoma might be avoided by giving potentially more efficacious plant mitogens like PHA as previously reported. The expanded disclosure of a collaborator's method suggesting superior potential for cancer cure involves a unique application of pokeweed mitogen that delivers various cellular and cytokine responses directly to the tumor.

Publication Types:

- Review
- Review, Tutorial

PMID: 10850356 [PubMed - indexed for MEDLINE]

---

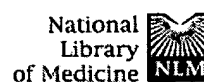
Display	Abstract	▼	Show:	20	▼	Sort	▼	Send to	Text	▼
---------	----------	---	-------	----	---	------	---	---------	------	---

[Write to the Help Desk](#)

[NCBI](#) | [NLM](#) | [NIH](#)

[Department of Health & Human Services](#)

[Freedom of Information Act](#) | [Disclaimer](#)



PubMed	Nucleotide	Protein	Genome	Structure	PMC	Taxonomy	OMIM	Books
Search PubMed	for						Go	Clear
Limits		Preview/Index		History		Clipboard		Details

Display: Abstract	Show: 20	Sort	Send to: Text
-------------------	----------	------	---------------

☐ 1: Exp Lung Res 1993 Nov-Dec;19(6):715-29

[Related Articles, Links](#)

Entrez  
PubMed

## **Pichinde virus-induced respiratory failure due to obstruction of the small airways: structure and function.**

**Schaeffer RC Jr, Bitrick MS Jr, Connolly B, Jenson AB, Gong F.**

PubMed  
Services

Department of Physiology, University of Arizona Health Sciences Center, Tucson 85724.

Related  
Resources

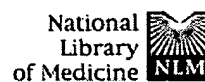
Respiratory distress that leads to death is seen in patients with Lassa fever. The development of this respiratory problem was studied using a Pichinde virus model (10(4) plaque forming units, IP, survival time 20 +/- 1 days) in strain 13 guinea pigs (n = 35, 229-353 g) of this lethal human contagious infectious disease. Extravascular lung water to bloodless dry lung weight (EVLW/BDLW) ratio showed a modest yet significant increase in animals 13 and 18-21 days postinoculation (PI). In contrast, residual lung blood and lung radioactive 125I-labeled human serum albumin activity index were elevated only in the 18- to 21-day group. These data are consistent with the progressive severity of perivascular edema, lymphocytic pneumonitis, and some alveolar protein between days 13 and 18-21 PI. Lymphocytic pneumonitis appeared to be distributed near most airways and was proportional to the degree of Pichinde virus antigen staining of alveolar macrophages, large mononuclear cells within the pulmonary vascular and extravascular spaces, and alveolar-capillary membranes. These findings suggest that lymphocyte recruitment to the lung reflects the Pichinde virus-induced cell-mediated immune response. Obstructed small bronchi with some luminal cell debris and hypertrophied epithelial cells were found associated with the areas of marked pneumonitis. The severe hypoxemia and modest anaerobic metabolism in association with marked tachypnea and normocapnia are consistent with small airway obstruction and wasted ventilation, since no change in arterial blood pressure, heart rate, hematocrit, hemoglobin, or blood volume was noted. These data suggest that Pichinde virus-induced respiratory failure was due to obstruction of the small airways with wasted ventilation in association with lymphocytic pneumonitis.

PMID: 8281916 [PubMed - indexed for MEDLINE]

Display: Abstract	Show: 20	Sort	Send to: Text
-------------------	----------	------	---------------

[Write to the Help Desk](#)  
[NCBI](#) | [NLM](#) | [NIH](#)  
[Department of Health & Human Services](#)  
[Freedom of Information Act](#) | [Disclaimer](#)

0



PubMed	Nucleotide	Protein	Genome	Structure	PMC	Taxonomy	OMIM	Books
Search	PubMed	for					Go	Clear
Limits		Preview/Index		History		Clipboard		Details

Display	Abstract	▼	Show: 20	▼	Sort	▼	Send to	Text	▼
---------	----------	---	----------	---	------	---	---------	------	---

☐ 1: Indian J Chest Dis Allied Sci 1999 Apr-Jun;41(2):115-9

[Related Articles, Links](#)

Entrez  
PubMed

## Dengue hemorrhagic fever (DHF) presenting with ARDS.

Sen MK, Ojha UC, Chakrabarti S, Suri JC.

Department of Pulmonary, Critical Care and Sleep Medicine, Safdarjung Hospital, New Delhi.

PubMed  
Services

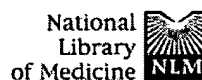
A patient of dengue hemorrhagic fever who progressed into acute respiratory distress syndrome (ARDS) is reported. The patient recovered after mechanical ventilation and supportive treatment.

PMID: 10437325 [PubMed - indexed for MEDLINE]

Display	Abstract	▼	Show: 20	▼	Sort	▼	Send to	Text	▼
---------	----------	---	----------	---	------	---	---------	------	---

Related  
Resources

[Write to the Help Desk](#)  
[NCBI](#) | [NLM](#) | [NIH](#)  
[Department of Health & Human Services](#)  
[Freedom of Information Act](#) | [Disclaimer](#)



PubMed	Nucleotide	Protein	Genome	Structure	PMC	Taxonomy	OMIM	Books
Search PubMed	<input type="checkbox"/> for						Go	Clear
Limits		Preview/Index		History		Clipboard		Details

Display	Abstract	<input type="button" value="v"/>	Show: 20	<input type="button" value="v"/>	Sort	<input type="button" value="v"/>	Send to	Text	<input type="button" value="v"/>
---------	----------	----------------------------------	----------	----------------------------------	------	----------------------------------	---------	------	----------------------------------

☐ 1: Rev Infect Dis 1983 Mar-Apr;5(2):346-52

[Related Articles, Links](#)

Entrez  
PubMed

## Delayed-type hypersensitivity: probable role in the pathogenesis of dengue hemorrhagic fever/dengue shock syndrome.

Pang T.

PubMed  
Services

The hypothesis presented proposes the involvement of a systemic form of a delayed-type hypersensitivity reaction in the pathogenesis of dengue hemorrhagic fever/dengue shock syndrome. It envisages the activation of sensitized T lymphocytes during a secondary infection by viral antigen present on the surfaces of mononuclear phagocytic cells. These antigen-activated T cells then release a variety of biologically active chemical mediators (lymphokines), which then produce the symptoms of shock and hemorrhage seen in cases of dengue hemorrhagic fever/dengue shock syndrome.

PMID: 6844807 [PubMed - indexed for MEDLINE]

Related  
Resources

Display	Abstract	<input type="button" value="v"/>	Show: 20	<input type="button" value="v"/>	Sort	<input type="button" value="v"/>	Send to	Text	<input type="button" value="v"/>
---------	----------	----------------------------------	----------	----------------------------------	------	----------------------------------	---------	------	----------------------------------

[Write to the Help Desk](#)  
[NCBI](#) | [NLM](#) | [NIH](#)  
[Department of Health & Human Services](#)  
[Freedom of Information Act](#) | [Disclaimer](#)

0